The Value of Dead Space Measurements in Mechanically Ventilated Patients

Respiratory failure is manifested by a derangement of arterial blood gases, and the relationship of arterial blood gases to the matching of ventilation ($\dot{V}$) to perfusion ($\dot{Q}$) in the lungs is critical to understanding the mechanisms of respiratory failure. Traditionally, $\dot{V}/\dot{Q}$ in the lungs is described in the 3-compartment model: normal $\dot{V}/\dot{Q}$, shunt, and dead space (V_D). A shunt exists in a pulmonary segment when where there is pulmonary arterial blood flow and no ventilation, that is $\dot{V} = 0/\dot{Q}$. Dead space may be manifested as low $\dot{V}/\dot{Q}$ or absolute, with no blood flow, that is $\dot{V}/\dot{Q} = 0$.

Although much of the management of patients in respiratory failure on artificial ventilation has focused primarily on imaging and respiratory mechanics, dead-space measurements have been shown to provide very useful information in patients with severe respiratory failure both for management and for prognosis. The concept of dead space was originally described by Bohr.[1] Dead space is divided into the normal anatomic dead space—this exists in the large and small airways, which normally do not participate in gas exchange—and the alveolar dead space when there is reduced or no blood flow to a given area of lung that is still receiving ventilation. The sum of anatomical V_D and alveolar V_D is referred to as the physiologic dead space. Traditionally, dead space is expressed as a fraction of the tidal volume (V_T), ie, physiologic V_D/V_T, but it may also be expressed as anatomical V_D/V_T or alveolar V_D/V_T.

The single-breath measurement of dead space originally described by Fowler[2] essentially measures anatomical V_D. By adapting this to the single-breath CO_2 expiration plot, Fletcher and colleagues[3] showed that both anatomical V_D and alveolar V_D could be calculated. The technique was further validated by Verschuren et al[4] in spontaneously breathing normal subjects and in patients with cardiopulmonary disease. An invasive technique for measuring dead space was described by Enghoff[5] as a modification of the Bohr equation,[1] utilizing arterial and expired P_{CO_2} measurements. This has become the standard technique for measurement of dead space. In contrast, the original Bohr equation[1] utilizes the alveolar P_{CO_2} rather than the arterial P_{CO_2}.

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Nuckton and colleagues[6] noted that the mean dead-space fraction, as measured with the Enghoff technique, was markedly elevated early in the course of ARDS and was significantly higher among subjects who died than among those who survived; furthermore, the dead-space fraction was an independent risk factor for death. Gattinoni et al[7] reported an increased survival at 28 d in subjects with ARDS who responded to prone positioning with a reduction of their P_{aCO_2} (ie, decreased V_D/V_T).

In subjects with ARDS, in whom dead space was measured with the Enghoff technique at days 1, 2, 3, and 6, Kallet et al[8] reported that a sustained elevation of V_D/V_T is characteristic of nonsurvivors, indicating that serial dead-space measurements may have prognostic value. Cepkova et al[9] noted that elevated V_D/V_T in 42 subjects with early acute lung injury on mechanical ventilation was associated with increased mortality and with fewer ventilator-free days; V_D/V_T measurement was compared to systolic pulmonary artery pressure, which was not predictive of outcome.

These study results were further validated by Siddiki et al,[10] who analyzed data from 2 prospectively collected acute lung injury/ARDS databases, which included data from 2,005 patients. The investigators noted an increase in percent mortality for every 10% increase in V_D/V_T in a dose-response fashion.[10]

The Enghoff calculation of dead space is affected by the level of shunt. In this issue of RESPIRATORY CARE, Tussman and associates[11] report a study comparing the measurement of dead space with the Bohr technique and with the Enghoff modification in a model of induced ARDS in pigs.[11] The investigators examined the effects of PEEP on the Bohr and Enghoff parameters under constant ventilation and stable hemodynamic conditions They observed that the V_D/V_T measured according to either technique was significantly elevated at all levels of PEEP. However, the alveolar V_D measured with the Bohr technique was not significantly increased at PEEP levels up to +15 cm H_2O,

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after which it increased at higher PEEP levels up to +30 cm H_2O. In contrast, the alveolar V_D calculated with the Enghoff technique was significantly elevated at all levels of PEEP, although there was a slight decrease at levels of +15 cm H_2O or higher. They attribute the changes in Bohr dead space to "stress" on the lungs, whereas the changes in the Enghoff dead space are attributed to the shunt effect.

As the authors recognize, however, there are a number of limitations to this study, primarily that the study design maintained the pigs at a hypercapnic, acidotic level, which could affect the measured dead space. In addition, their attribution of shunt as the primary cause of the very high alveolar–arterial oxygen difference values up to 51 mm Hg is arguable and is not supported by previously published data. Nevertheless, the value of dead space measurement in patients with ARDS is well established, and it is clear that measurement of dead space with the standard Enghoff or volumetric capnography techniques provides very useful information about the underlying gas-exchange abnormality and prognosis in patients on mechanical ventilation.

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